Tendonitis (bowed tendon) 9/8/08

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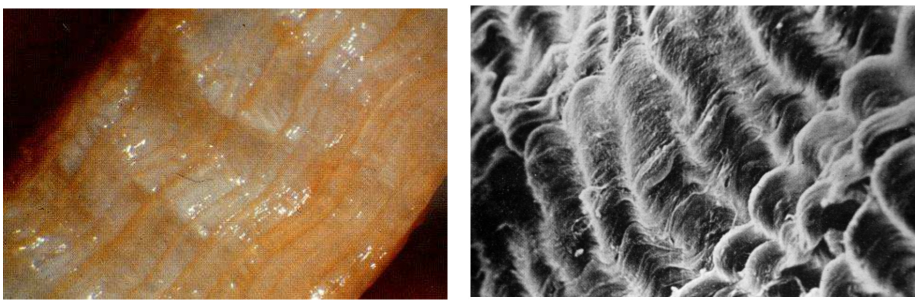
Tendonitis of the superficial digital flexor tendon (SDFT) is most common in the forelimbs of racing Thoroughbreds, quarter horses, and sport horses. In racing breeds, particularly the Thoroughbred, it is considered a career-compromising injury and the outlook for a return to performance is pessimistic. One study evaluating injuries sustained during racing at race tracks in the United States found that out of 1,100 reports, tendonitis of the SDFT and suspensory ligament (SL) desmitis/rupture were the most common. In another report, a 30% incidence of SDFT tendonitis was found in horses in training in the United Kingdom and continental Europe. Injury to the superficial digital flexor tendon of the hind limb is uncommon in all breeds, but does occur to some extent in the Standardbreds.

Hemorrhage and varying degrees of tearing of the collagen fibrils occur within the affected tendon. Necrosis of the torn fibers may develop as a result of disruption in the blood supply. When sufficient fiber disruption occurs, severe swelling plus tendon lengthening may be seen, and this is generally manifested by a dropped fetlock. Adhesions develop between the tendon and surrounding tissues, and there is a varying amount of fibrosis that develops in the chronic stage. It is this inflammatory tissue deposition which results in the bowed appearance. The primary injury heals by fibrosis and the prognosis for return to racing lies between 30% - 65%.

Anatomy and composition

Tendons form a continuation of the collagenous fascial framework that surrounds and supports the muscle and its fibers. When the surface of the tendon is illuminated with low-angle incident light, a light and dark banding is apparent, indicating an undulating surface. This is caused by the presence of a planar wave-form or crimp which contributes to the complex mechanical behavior of tendons.

Fig. 1 A&B illustrating crimp/wave pattern.



Under light microscopy, tendons are composed of an extracellular matrix (predominantly parallel collagen fibers) interspersed with tendon cells. Three cell types are observed.

It is not known if these cells have specific roles in tendon metabolism or merely represent different stages in maturation of tenocytes. The cells are responsible for the production and maintenance of the surrounding extracellular matrix. No data for the rate of collagen turnover is available for the horse. In adult horses, tendon fibrils vary in their size–small, medium, and large. The tendon fibrils are predominantly of small diameter in the early stages of repair after injury and change toward the larger adult size as the scar matures. The small fibrils are believed to be mechanically weaker that the large ones, so healing tendons are weaker. Strength increases as the fibril diameter and the number of stable chemical crosslink's increase. A considerable time is required for a tendon to reach maximum strength after injury. There is some evidence that fibril aggregation may be inhibited by high hyaluronate concentration and that fibril maturation may be controlled by glycosaminoglycan.

The cells produce and maintain the extracellular matrix, which is composed of collagen, elastin, proteoglycans, and glycoproteins. Collagen (primarily) and elastin (to a lesser degree) provide the tendons tensile strength.

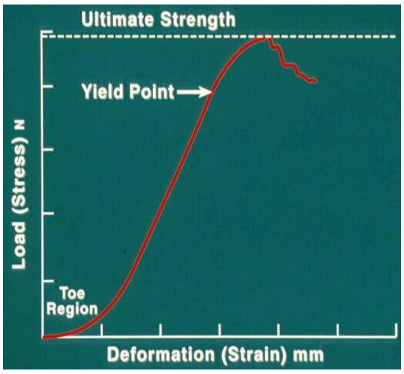
Collagen types are—type I (95%) and type III and V (5%). Type I and II collagen molecules are very similar and both form fibrils with the same banding periodicity. The diameter of the fibrils differs, however, with type III being small diameter and type I forming fibrils that can have larger diameters. The relative proportions of these two types of collagen therefore determine the mechanical properties of the tissue.

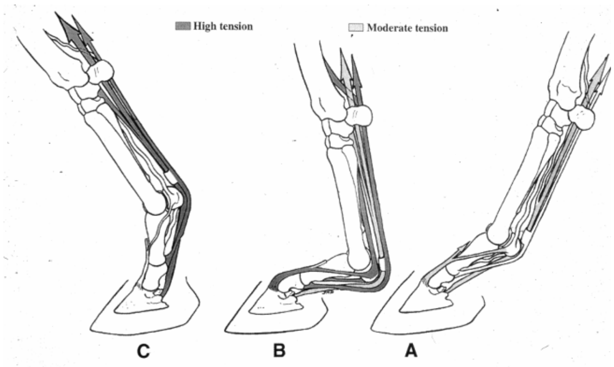
Functional and biomechanical properties

Flexor tendons, as their name implies, flex the digit as the limb is in the swing phase and are under high tensile stress as in the stance phase. The elastic energy stored in the flexor tendons under stress is used in the subsequent stride to reduce energy requirements and increase efficiency. To maximize the energy stored, the tendons elongate to a point close to failure. This may account for the relatively high incidence of injury during athletic activity.

The physical properties of tendons include high tensile strength, flexibility, and elasticity. Tendon is almost a perfect elastomer up to 3% strain. This is the point where crimp angles straighten and return to a normal angle. Beyond 3% strain the tendon becomes more resistant to elongation and a linear stress strain relationship develops until the yield point (disengagement of most microfibrils) is reached. Following is structural failure at strains of 12%-20% (Fig 2 A). Since the crimp in the central region of the tendon straightens before the periphery, in mature horses, the central region is predisposed to injury. When cadaver tendons are stretched to rupture, the central fibers fail first. The strain magnitudes in the SDFT in galloping Thoroughbred horses are very close to yield and failure. *In vivo* studies indicate that the SDFT bears the load in the early part of the stance phase of gait, before the load is shared with the deep digital flexor tendon (DDFT). The rate of rise in load also is greatest in the superficial flexor tendon. Peak loads occur in the deep digital flexor later in the gait cycle, and the rate of loading is lower, which may account for the low incidence of lesions in that tendon (Fig 2B).

Fig 2 A&B Notes:



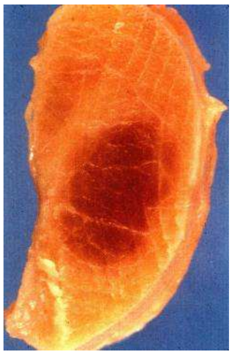


Mechanisms of injury and repair

Exercise-induced hyperthermia

In vivo recordings have shown that the central core temperature of the SDFT rises to around 45℃ during a 7-minute gallop, whereas the s urface temperature remains some 10℃ lower. This central core thermal effect may r esult in impaired metabolism of tenocytes or cell death. Discoloration of the central core of the SDFT has been observed in Thoroughbred racehorses destroyed for reasons other than tendon injury (Fig 3).

Fig 3 Notes:



Although these areas of discoloration have not been associated with histopathologic abnormalities, biochemical analysis has shown significant elevation in type III collagen and an increase in the GAG levels. The finding that central core temperature can reach 45℃ during exercise suggests a possible cause for cell damage and an alteration of matrix synthesis.

Reperfusion injury

The cyclical loading of tendons may result in ischemia during the period the tendon is under maximum tension. When relaxed, a reperfusion may occur in which superoxides may be formed. Free oxygen radicals are toxic to tissues and cause local damage to cells and matrix synthesis.

Repair
After tendon injury, the repair process follows the same pattern as seen in other tissues. Initial fibriller rupture is followed by the formation of an intratendinous hematoma, with swelling and inflammation. Following inflammatory process, the damaged tissue is removed by scavenger cells, after which a scar is formed. In the new scar tissue, the collagen initially is immature and arranged in a haphazard architecture, comprising predominantly type III collagen with small-diameter fibrils. Maturation then follows, in which the fibril diameters increase, as do the stable chemical cross-links, together with the proportion of type I collagen. The initial haphazard arrangement of collagen fibers gradually changes to a more parallel alignment as functional tendon loading is restored. This process takes weeks to months to reach completion, and even then the original strength of the tendon is not restored.

Predisposing causes
The various predisposing causes that have been incriminated are largely anecdotal and they include:

1. Inadequate training and muscle fatigue at the end of the long race results in poor muscle response and loss of tendon stabilization.

2. Abnormal angulations of the fetlock associated with muscle weakness or conformation increases the stresses on the tendon.

3. Uneven and slippery ground or sudden turns may load one side of the tendon disproportionately.

4. Excessive pastern slope, improper shoeing. (Short shoeing, shoeing with long toe/low heel, a low hoof angle, toe grabs, club feet).

5. Muddy tracks increase the workload on the tendon and tight fitting bandages or boots have also been implicated.

6. Incoordination and disproportion between the body weight and tendon strength.

Clinical signs and diagnosis
In the acute phase there is diffuse swelling with heat and pain on palpation. An exception to this is when the deep digital flexor tendon alone is involved in the digital sheath. Minimal swelling may be present. The degree of lameness largely depends on the severity of the injury. Major disruption in the tendon fibers or stretching of the tendon will result in a dropped fetlock.

The chronic stage is manifested by fibrosis of the metacarpal/metatarsal region and hard swelling of the palmar and plantar aspect. Some acute inflammatory changes may still be present, depending on the stage of healing or if the injury has occurred. The horse may be sound at a walk and trot but become lame with hard work. Anular ligament constriction or desmitis may be present in association with a chronic low bow at the level of the fetlock (fig 4).

Fig 4. Examples of chronic low "bows" with concurrent annular ligament constriction

Although the diagnosis is made easily from the clinical signs, ultrasound examination is most important for defining the nature and extent of the tendon injury and the prognosis (fig 5). Ultrasound examination is also used to monitor the progress of healing, directing then the return to performance. Radiographs should be taken to identify calcified densities, fractured splints or other bone and joint abnormalities. Ancillary diagnostic imaging includes thermography and tendonography.

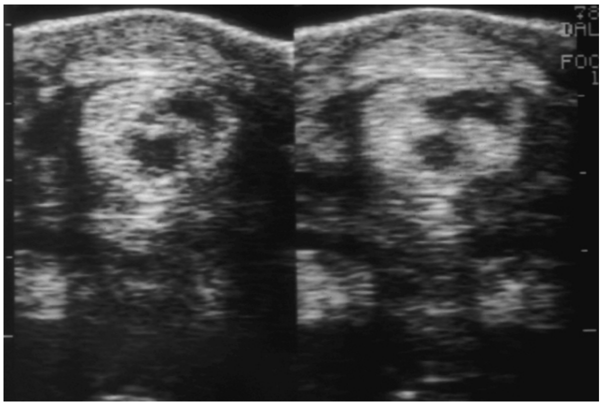


Fig 5 Illustrating core lesions in the DDFT

Treatment:

The goals of therapy for acute tendonitis are to decrease inflammation and swelling, minimize scar tissue formation, and promote restoration of normal tendon structure and function as soon as possible.

Acute (within 48 hours of injury)

Treatment is aimed at reducing the swelling and inflammation with pressure, cold treatments and anti-inflammatory drugs. Cold therapy should be administered for at least 24 hours or until the acute inflammation subsides, along with absolute confinement for at least 48 hours.

Cold therapy

Cold therapy can be administered as hydrotherapy, with ice or in an ice water slurry (best). Studies have shown that an ice water slurry deep cools the tissues more rapidly than cold water or ice alone. Cold therapy effectively inhibits inflammation. It constricts injured vessels, slows hemorrhage, reduces the effect of inflammatory mediators, reduces protein loss from vessels, and provides some relief of pain. Application of cold for 20 to 30 minutes 3 to 4 times per day is best. Short periods of cold therapy have been shown to have a sustained cooling effect. Prolonged application of cold (> 30 minutes) should be avoided because it may result in reflex vasodilation. Bandaging the lower limb with firm and even pressure also helps increase interstitial fluid pressure and counteract the Starling forces that promote fluid loss from the vessels.

Race plates or shoes with grabs are removed and the hoof should be trimmed and balanced to reduce any abnormal forces. An excessively long toe or high heel should be corrected to decrease tendon stress. Raising the heel was advocated to decrease stress on the superficial flexor tendon, but this theory has been refuted.

Nonsteroidal anti-inflammatory drugs (NSAIDs)
NSAID's drugs are used to reduce prostaglandin-mediated inflammation and to reduce the release of thromboxane from injured tissue. Banamine may be administered first because of its rapid onset followed by phenylbutazone which is administered until inflammation subsides.

Corticosteroids
Corticosteroids are potent anti-inflammatory drugs but they are generally not used for treatment of tendonitis because they can delay healing by inhibiting fibroplasia, collagen and glycosaminoglycan synthesis, both of which are important for repair. Injection of corticosteroids into normal tendons has been shown to cause tendon fiber necrosis, fibrocyte death and dystrophic calcification which decreased tensile strength for up to one year. Additionally, some steroids are implicated in the development of laminitis.

DMSO
The use of DMSO is rational for the treatment of acute inflammation. It not only has fluid disbursing properties but it also has the ability to scavenge superoxide radicals which can further damage the affected tendon. The vasodilatory properties of DMSO also may be of benefit in ischemic conditions. Although DMSO is considered to be of low toxicity, a study done in rats demonstrated that topical DMSO decreased the tensile strength of normal tail tendons, with a maximum decrease in strength of 20% after 7 days of use.

Subacute (generally 48 hours after injury)
The treatment of subacute tendonitis is aimed at stopping spread of inflammation into the normal tendon, to reverse the effects of inflammation and to facilitate the repair process.

Heat and cold therapy
Alternating temperature (cold and heat) therapy can begin after the acute inflammation subsides, usually 48 hours after injury, and is continued for four to six days. The warm interval should be three times as long as the cold treatment and this can be done at least three to four times a day. Warm therapy is continued after six days to improve

circulation and healing. Topical medication can be used for prolonged warm treatment. Pressure bandaging is continued in some cases out to three weeks.

Hyaluronic acid (HA)
Hyaluronic acid injected peritendinous in man has been shown to reduce adhesion formation. Its use in the horse has been questioned since some studies have not found a clear benefit. However, tendons injured within their sheath may benefit from injection of HA directly into the sheath. In one study, instillation of hyaluronic acid within the tendon sheath surrounding a collagenase-injured tendon improved the histologic appearance and maturation of the repair tissue at eight weeks after injury compared with controls. It was thought that intralesional injection of HA may be of benefit. However one large clinical trial found no benefit to intralesional injection of HA compared to controls.

Polysulfated glycosaminoglycan (PSGAG)
Polysulfated glycosaminoglycan has been shown to inhibit lysosomal enzymes and curtail the inflammatory process. They may be important in stimulating collagen synthesis. Clinical evaluation of a small number of horses with SDFT tendonitis indicated that treatment with PSGAG was more successful than conservative or laser therapy, but fewer than 50% of the horses were able to race and there was a higher injury recurrence rate with polysulfated glycosaminoglycan. PSGAG, one injection 1 mg/kg intramuscularly every 4 days for 4 weeks has been recommended. In the case of a severe lesion, the initial injection can be made into the lesion.

In a study evaluating different treatments for SDF tendonitis, there was no difference in the incidence of recurrence of tendonitis in horses treated conservatively compared with those treated intralesional with HA or PSGAG or treated systemically with PSGAG.

Heparin
Heparin's anticoagulant activity has been used successfully to decrease abdominal adhesion formation in horses; it also has enjoyed anecdotal success in the treatment of tendonitis. Heparin also may play a role in angiogenesis, but no studies have identified a clear benefit in treating tendonitis.

β-aminoproprionitrile (BAPN)
β-aminoproprionitrile (BAPN) is a lathyrogen that acts by irreversibly binding to lysyl oxidase which, in turn, inhibits the covalent cross linking of connective tissue proteins, elastin, and collagen. A clinical study in horses with tendonitis indicates that intralesional injection of BAPN results in more rapid improvement in the ultrasonographic appearance of an injured tendon than other methods of therapy. Affected tendons were injected every other day for 10 days and ultrasonographically evaluated during a 19-week period. The sonograms were evaluated by assigning a severity rating to the tendon lesions. At all time periods, the BAPN-treated tendons had a greater reduction in severity score than tendons treated by various other methods. Recent studies have cast doubt on the efficacy of BAPN treatments and in some an

adverse effect was seen (Dahlgren et al: AJVR 2002, Yammato et al: J Comp Patho, 2002). ***BAPN has been taken off the market in North America***.

Insulin like growth factor (IGF 1)
Insulin like growth factor – 1 has been shown to increase cell migration, stimulate extracellular matrix production and cell proliferation. Controlled trials have shown a clear benefit to its use. Better ultrasound scores, histologic scores and tensile strength gain has been observed. At the time of this writing IGF is not commercially available.

Stem cells
Injection of; bone marrow aspirates, ex-vivo cultured bone marrow stem cells and extracellular matrix (ACell) to promote host stem cells to heal the tendon site more rapidly with tissue that resembles the normal tendon structure is being evaluated at the time of this writing.

One study evaluating the effects of injecting bone marrow derived stem cells into the superficial digital flexor tendon of horse suffering tendonitis found 82% (9/11) stem cell treated horses were racing at 2 years post-injury and 100% (15/15) standard treatment horses re-injured their SDFT by 12 mo post-injury. (Pacini S et al: Tissue engineering, 2007)

Plasma rich platelet gel (PRP)
Injection of PRP is being used to enhance healing of tendons suffering from tendonitis and ligaments suffering from desmitis. Several systems are available, all of which have special claims as to the ease of collection, increased harvest of platelets and cost advantage. To my knowledge no studies have clearly proven the superiority of PRP compared to Stem cell injections in the healing rates of tendons and ligaments. A few of the companies that have collection systems are listed: Magellan Platelet Gel), Harvest SmartPReP™ 2 and Biomet GPS III. Theoretically the combination of PRP, injected first followed by the injection of Stems cells should provide superior healing compared to either, used alone.

Electrical stimulation
Many of the proposed physiologic effects of electrical stimulation on tissues would seem to be of benefit to tendon repair, but evidence is lacking to support its use in tendon injuries. Several studies in fact refute its use. One study found that daily electromagnetic therapy did increase the vascularity of surgically created superficial digital flexor tendon defects but repair tissue maturation and collagen type transformation actually were delayed by the treatment in samples collected at 8 and 12 weeks after surgery. Another study using direct current also found no therapeutic benefits on tendon healing.

Shock wave
Treatment with extracorporeal shock waves is being evaluated at this time. Treatment regimes and outcomes have been variable. Anecdotal comments indicate it may be of benefit. Some studies have found neurovascular damage.

Low-level laser
Low-level laser therapy, also called soft or cold laser therapy, has been used in the treatment of various soft tissue and skeletal disorders in the horse. A specific energy range is used to induce a biologic effect. The therapeutic effects are believed to include; increase of protein synthesis, improved blood flow, lymphatic regeneration, pain relief, improved cellular phagocytic activity, fibroblast proliferation, and collagen production. These biologic effects depend on the frequency, wavelength, and energy, tissue type, and the duration and frequency of application.

In one study, laser therapy for SDF tendonitis resulted in a 50% return to training, 30% return to racing, and 20% injury recurrence rate in National Hunt racehorses. These results were not as favorable as those treated conservatively. Another clinical study found that 66% of Standardbred racehorses were able to return to racing following laser treatment of tendonitis. Others found no benefit from laser irradiation of skin and tendon wounds in horses.

Counter irritation
Counter irritation has the potential to increase blood flow to the injured tendon, but the inflammation created by this treatment would increase fibrous tissue deposition in and around the tendon. The additional scar tissue may give strength to the tendon by increasing its functional cross-sectional area but would adversely affect the viscoelastic properties of the tendon, which ultimately would decrease function. One study found that pin firing during the chronic states of tendonitis prolongs healing of the tendon and results in further damage to the tendon. There therefore appears to be no place for counter irritation in the treatment of tendonitis, especially if athletic function is a goal of treatment.

Surgical treatment
Surgical treatment includes percutaneous tendon slitting, radial (superior) check ligament desmotomy and digital annular ligament desmotomy.

Percutaneous tendon splitting (PTS)
It is hypothesized that PTS creates a communication between the tendon core and the surrounding peritendinous tissues. These openings promote more rapid resolution of inflammatory edema and expedite revascularization and collagen production within the area of injury. It would appear that tendon splitting during the early stage of tendonitis has beneficial effects on the early repair process. PTS is done with a double edged tenotome or a #11 scalpel blade. Ultrasound guidance is recommended

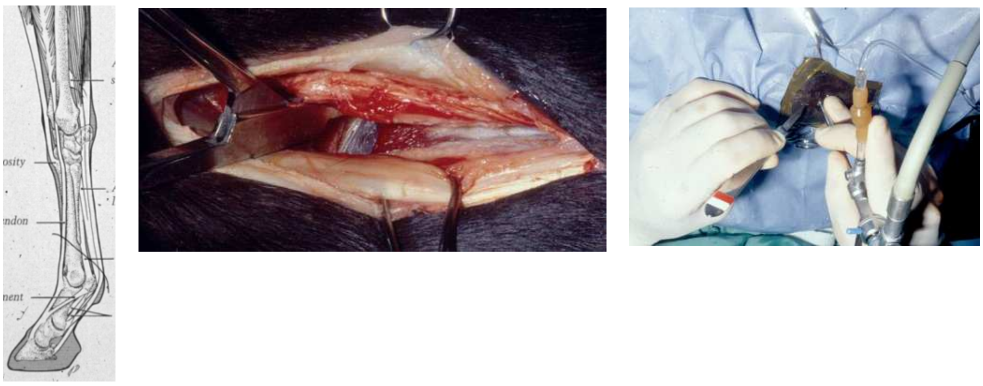
A clinical study demonstrated that the splitting of acutely to subacutely injured tendons that contained core lesions resulted in reduction in lesion size, tendon diameter, and lesion grade within 8 to 12 days after surgery. Long-term follow-up documented a decrease in tendon diameter and, therefore, most likely a reduction in fibrous tissue deposition. Another study found a reduction in lesion size of 44% and mean decrease in lesion grade of 0.9 by day 10 following ultrasonographically guided tendon splitting.

Eighty-one percent (81%) of the horses were able to return to performance and 68% competed at the same level following surgery. This is impressive considering that these grade 3 or 4 tendonitis lesions involved up to 80% of the cross-sectional area of the tendon. In a controlled study using a collagenase-induced tendonitis, there was significant improvement in the ultrasonographic grade of lesions in tendons that underwent splitting; there was earlier revascularization of the lesions and superior-quality repair tissue at 4 and 8 weeks after surgery. Presently, this procedure is recommended for cases of tendonitis with anechoic core lesions. To be most effective, PTS should be done within 2 to 3 days after injury, although some benefit may be appreciated out to 2 weeks.

Radial check ligament desmotomy (RCLD)

Radial (superficial) check ligament desmotomy is generally done as early as possible after injury, although some benefit is seen in cases done at a later time. It has been proposed that cutting the check ligament to the superficial digital flexor tendon lengthens the musculotendinous unit (fig 6A). Even though it is likely that the RCL heals after transection, it does so in an elongated fashion. In theory, this would increase the elastic limit of the damaged tendon, thus reducing the effect of intrinsic loss of elasticity found in healed scarred tendons and minimizes the chances of reinjury of the torn tendon. It can be done by surgical incision (fig 6B) or by tenoscopy (fig 6C).

Fig 6 A,B&C



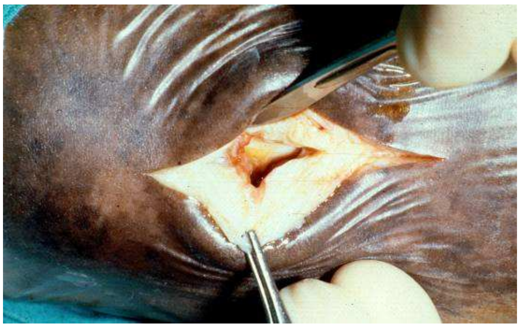
In a study evaluating the use of RCLD in the treatment of tendonitis in 137 Thoroughbred racehorses, they found that 71% of the horses were able to train and race after surgery and 51% had more than five starts after the surgery, but average earnings dropped in 58% of the horses. Only 19% reinjured the tendon, which appears to be significant considering that the majority of the horses in this study were experiencing at least their second episode of tendonitis at the time of the surgery. In this study, a mean time from surgery to the first start was 353 days. In a smaller separate study evaluating the long-term effects of RCLD and other treatments, 53% flat racehorses, 58% steeplechasers, 69% Standardbred racehorses, and 73% hurdlers competed in five or more starts after surgery. These results compare favorably with results from conservative management of tendinitis where approximately 50% of the horses returned to competition, but in a lower class, and recurrence of tendinitis was common (48%). It appears from these studies that radial check ligament desmotomy increases a horse's chances of returning to competition and decreases the incidence of reinjury compared with other methods of therapy.

Another study done on 124 Thoroughbred racehorses evaluating the effects of RCLD (31 horses) versus nonsurgical treatment (NST) (93 horses) found no benefit to RCLD versus NST in preventing recurrent or new injuries. Recurrent or new injuries occurred in 66% NST horses and 78% RCLD horses. Horses undergoing RCLD were 5.5 times as likely to develop SLD as NST horses. Also, no significant association was found between length, type or cross-sectional area of the ultrasonographic defect in the SDFT between treatment groups.

Annular ligament desmotomy (ALD)

Annular ligament desmotomy is usually combined with RCLD to treat distal tendinitis of the SDFT. As the SDFT enlarges, the gliding function becomes impeded by the inelastic annular ligament. As time passes, the annular ligament becomes thickened further limiting the gliding function and possibly the blood supply to the SDFT. Once the SDFT function is impeded by the annular ligament, continued inflammation and swelling often exacerbate the original lameness. Severing the AL provides immediate decompression and improves the gliding function of the injured tendon. Pain relief is often immediate and a reduction in the size of the injured tendon is usually seen between five to ten days after surgery. The procedure can be done arthroscopically (difficult with an enlarged tendon) or by incision (my preference) (fig 7).

Fig 7 Notes:



Rehabilitation program

Research indicates that it takes some six months for tendon collagen type, cellularity, and fiber crimp pattern (all of which affect tendon strength) to return to normal after injury. Therefore, it is not surprising that six months of restricted exercise is recommended in cases of tendinitis treated. Based on ultrasonographic findings and clinical experience with some 2,800 cases of tendon and ligament injuries, Gillis (1997) has outlined a rehabilitation protocol. The protocol takes into account the clinical findings, ultrasonographic changes, and the response to therapy at subsequent examinations over a 360 day period. It was found that it usually takes eight to nine months of rest and controlled exercise for horses to return to their previous workload. The premise for the use of controlled exercise is to initially reduce inflammation, maintain gliding function, and improve healing.

Aftercare for surgically treated and nonsurgically treated consists of two weeks of absolute stall confinement followed by four weeks of stall rest with hand walking 10 to 15 minutes twice daily. Thereafter, a progressive hand walking exercise program is instituted for an additional six weeks. The tendon is reevaluated clinically and ultrasonographically at three month intervals until healing is complete. Training should progress slowly and the tendon should be closely evaluated for evidence of inflammation because each subsequent injury results in further tendon fibrosis.

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